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Search Results -

Term	Documents
"HER.BETA".DWPI,TDBD,EPAB,JPAB,USPT,PGPB.	1
HER.BETAS	0
HER.	0
HER.S	0
BETA.	0
"BETA.S".DWPI,TDBD,EPAB,JPAB,USPT,PGPB.	352
(("HER." ADJ "BETA.") OR "HER.BETA").USPT,PGPB,JPAB,EPAB,DWPI,TDBD.	1

Database: US Patents Full-Text Database; US Pre-Grant Publication Full-Text Database; JPO Abstracts Database; EPO Abstracts Database; Derwent World Patents Index; IBM Technical Disclosure Bulletins

Refine Search: her.beta or her. beta. Clear

Search History

Today's Date: 5/18/2001

DB Name	Query	Hit Count	Set Name
USPT,PGPB,JPAB,EPAB,DWPI,TDBD	her.beta or her. beta.	1	L4
USPT,PGPB,JPAB,EPAB,DWPI,TDBD	herbeta or her-beta or her beta	4	L3
USPT,PGPB,JPAB,EPAB,DWPI,TDBD	human estrogen receptor	252	L2
USPT,PGPB,JPAB,EPAB,DWPI,TDBD	estrogen receptor	1674	L1

FILE 'MEDLINE'
FILE 'JAPIO'
FILE 'BIOSIS(C) 2001 BIOSIS(R)

FILE 'SCISEARCH'
FILE 'WPIDS'
FILE 'CAPLUS'
FILE 'EMBASE'
=> s estrogen receptor#

5 FILES SEARCHED...
L1 79174 ESTROGEN RECEPTOR#

=> s l1 and (her beta or her-beta or herbeta or her.beta or her. beta.)

5 FILES SEARCHED...
L2 43 L1 AND (HER BETA OR HER-BETA OR HERBETA OR HER.BETA OR HER. BETA.)

=> dup rem l2

PROCESSING COMPLETED FOR L2
L3 13 DUP REM L2 (30 DUPLICATES REMOVED)

=> d l3 ibib abs 1-13

L3 ANSWER 1 OF 13 WPIDS COPYRIGHT 2001 DERWENT INFORMATION LTD
ACCESSION NUMBER: 2001-168581 [17] WPIDS
DOC. NO. NON-CPI: N2001-121530
DOC. NO. CPI: C2001-050401
TITLE: Determination of the interaction of a substance for
investigation and diagnosis of hormonal disorders using
an optically labelled receptor protein.
DERWENT CLASS: B04 D16 S03
INVENTOR(S): KATO, N; SAKAMOTO, H
PATENT ASSIGNEE(S): (OLYU) OLYMPUS OPTICAL CO LTD
COUNTRY COUNT: 8
PATENT INFORMATION:

PATENT NO	KIND	DATE	WEEK	LA	PG
WO 2001007919 A1		20010201	(200117)*	JA	75
RW: DE FI FR GB NL SE					
W: JP US					

APPLICATION DETAILS:

PATENT NO	KIND	APPLICATION	DATE
WO 2001007919 A1		WO 2000-JP4930	20000724

PRIORITY APPLN. INFO: JP 2000-163476 20000531; JP 1999-209860
19990723; JP 2000-163475 20000531
AN 2001-168581 [17] WPIDS
AB WO 200107919 A UPAB: 20010328
NOVELTY - Examination of the interaction of a test substance with a
hormone receptor protein is new.
DETAILED DESCRIPTION - The method comprises:
(a) the test substance is contacted with a hormone receptor protein
labelled with a marker which generates an optical signal under conditions
in which the receptor protein can bind to a ligand and after binding
undergo a change of state so as to alter the properties of the optical
signal;
(b) the optical signal is detected under these conditions; and
(c) the optical signal produced is compared with that generated in
the absence of the test substance, to show whether the test substance is
interacting with the hormone receptor protein.
INDEPENDENT CLAIMS are also included for:
(1) genes encoding the labelled hormone receptor protein;
(2) vectors containing the genes;
(3) host cells transformed by the vectors; and
(4) the labelled hormone receptor protein.
USE - Investigation and diagnosis of hormonal disorders especially
those of sex hormones such as suppression of ovulation.
DESCRIPTION OF DRAWING(S) - The drawing shows the fluorescence
correlation spectroscopy traces of a green fluorescent protein/human
estradiol receptor beta fusion protein at the time of addition of
estradiol and after 45 minutes from addition - the shift in the trace
shows that interaction of the hormone and receptor is taking place.
(Drawing includes non-English language text).
Dwg.14/14

L3 ANSWER 2 OF 13 SCISEARCH COPYRIGHT 2001 ISI (R) DUPLICATE 1
ACCESSION NUMBER: 2001:165025 SCISEARCH
THE GENUINE ARTICLE: 401HB
TITLE: Unique protein determinants of the subtype-selective
ligand responses of the ***estrogen*** ***receptors*** (ER alpha and ER beta) at AP-1 sites
AUTHOR: Weatherman R V; Scanlan T S (Reprint)
CORPORATE SOURCE: Univ Calif San Francisco, Dept Pharmaceut Chem, San
Francisco, CA 94143 USA (Reprint); Univ Calif San
Francisco, Dept Cellular Mol Pharmacol, San Francisco, CA
94143 USA
COUNTRY OF AUTHOR: USA
SOURCE: JOURNAL OF BIOLOGICAL CHEMISTRY, (9 FEB 2001) Vol. 276,
No. 6, pp. 3827-3832.
Publisher: AMER SOC BIOCHEMISTRY MOLECULAR BIOLOGY INC,
9650 ROCKVILLE PIKE, BETHESDA, MD 20814 USA.
ISSN: 0021-9258.
DOCUMENT TYPE: Article; Journal
LANGUAGE: English
REFERENCE COUNT: 38
ABSTRACT IS AVAILABLE IN THE ALL AND IALL FORMATS
AB The two subtypes of human ***estrogen*** ***receptor*** , alpha
(hER alpha and beta (***hER*** ***beta***), regulate transcription
at an AP-1 response element differently in response to estradiol and the
anti-estrogens tamoxifen and raloxifene. To better understand the protein
determinants of these differences, chimeric and deletional mutants of the
N-terminal domain and the F region of ER alpha and ER beta were made and
tested in transient transfection assays at the classical estrogen response
element (ERE) site as well as at an AP-1 site. Although the same regions
on each receptor subtype appeared to be primarily responsible for
estradiol activation at an ERE and in HeLa cells, major differences
between ER alpha and ER beta mutants were seen in the estrogen and
anti-estrogen responses at an AP-1 site. This differential ligand response
maps to the N-terminal domain and the F region. These results suggest that
different estrogenic and anti-estrogenic ligands use different mechanisms
of activation and inhibition at the AP-1 site. In contrast to previous
studies, this work also shows that many of subtype-specific responses are
not transferred to the other subtype by swapping the N-terminal domain of
the receptor. This implies that there are other unique surfaces presented
by each subtype outside of the N-terminal domain, and these surfaces can
play a role in subtype-selective signaling. Together, these data suggest a
complex interface between ligand, response element, and receptor that
underlies ligand activation in estrogen signaling pathways.

L3 ANSWER 3 OF 13 SCISEARCH COPYRIGHT 2001 ISI (R) DUPLICATE 2
ACCESSION NUMBER: 2001:294637 SCISEARCH
THE GENUINE ARTICLE: 416UG
TITLE: Interaction of phytoestrogens with ***estrogen***
receptors alpha and beta
AUTHOR: Morito K; Hirose T; Kinjo J; Hirakawa T; Okawa M; Nohara
T; Ogawa S; Inoue S; Muramatsu M; Masamune Y (Reprint)
CORPORATE SOURCE: Kanazawa Univ, Fac Pharmaceut Sci, Dept Mol & Cellular
Biol, 13-1 Takara Machi, Kanazawa, Ishikawa 9200934, Japan
(Reprint); Kanazawa Univ, Fac Pharmaceut Sci, Dept Mol &
Cellular Biol, Kanazawa, Ishikawa 9200934, Japan; Fukuoka
Univ, Fac Pharmaceut Sci, Lab Pharmacognosy, Fukuoka
8140180, Japan; Kumamoto Univ, Fac Pharmaceut Sci, Lab Nat
Med, Kumamoto 8620973, Japan; Univ Tokyo, Grad Sch Med,
Dept Geriatr Med, Bunkyo Ku, Tokyo 1138655, Japan; Saitama
Med Sch, Dept Biochem, Moroyama, Saitama 3500451, Japan
COUNTRY OF AUTHOR: Japan
SOURCE: BIOLOGICAL & PHARMACEUTICAL BULLETIN, (APR 2001) Vol. 24,
No. 4, pp. 351-356.
Publisher: PHARMACEUTICAL SOC JAPAN, 2-12-15-201 SHIBUYA,
SHIBUYA-KU, TOKYO, 150, JAPAN.
ISSN: 0918-6158.
DOCUMENT TYPE: Article; Journal
LANGUAGE: English
REFERENCE COUNT: 37
ABSTRACT IS AVAILABLE IN THE ALL AND IALL FORMATS
AB The human ***estrogen*** ***receptor*** (hER) exists as two
subtypes, hER alpha and ***hER*** ***beta*** , that differ in the
C-terminal ligand-binding domain and in the N-terminal transactivation
domain. In this study, we investigated the estrogenic activities of soy
isoflavones after digestion with enteric bacteria in competition binding
assays with hER alpha or ***hER*** ***beta*** protein, and in a
gene expression assay using a yeast system. The estrogenic activities of
these isoflavones were also investigated by the growth of MCF-7 breast
cancer cells.
Isoflavone glycoside binds weakly to both receptors and ***estrogen*** ***receptor*** -dependent transcriptional expression
is poor. The aglycones bind more strongly to ***hER*** ***beta***
than to hER alpha The binding affinities of genistein, dihydrogenistein
and equol are comparable to the binding affinity of 17 beta -estradiol,
Equol induces transcription most strongly with hER alpha and ***hER***
beta . The concentration required for maximal gene expression is
much higher than expected from the binding affinities of the compounds,
and the maximal activity induced by these compounds is about half the
activity of 17 beta -estradiol, Although genistin binds more weakly to the
receptors and induces transcription less than does genistein, it
stimulates the growth of MCF-7 cells more strongly than does genistein.

L3 ANSWER 4 OF 13 MEDLINE
DUPLICATE 3
ACCESSION NUMBER: 2001204445 MEDLINE
DOCUMENT NUMBER: 21111546 PubMed ID: 11158716
TITLE: Hydroxylated benzo[a]pyrene metabolites are responsible for
in vitro ***estrogen*** ***receptor*** -mediated
gene expression induced by benzo[a]pyrene, but do not
elicit uterotrophic effects in vivo.
AUTHOR: Fertuck K C; Matthews J B; Zacharewski T R
CORPORATE SOURCE: Department of Biochemistry and Molecular Biology, Michigan
State University, Lansing, Michigan 48824, USA.
SOURCE: TOXICOLOGICAL SCIENCES, (2001 Feb) 59 (2) 231-40.
Journal code: CZ1; 9805461. ISSN: 1096-6080.
PUB. COUNTRY: United States
Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200104
ENTRY DATE: Entered STN: 20010417
Last Updated on STN: 20010417
Entered PubMed: 20010222
Entered Medline: 20010412
AB The estrogenic activities of benzo[a]pyrene (B[a]P) and 10 metabolites (1,
3-, 7-, and 9-hydroxy-B[a]P; 4,5-, 7,8-, and 9,10-dihydrodihydroxy-B[a]P;
and 1,6-, 3,6-, and 6,12-B[a]P-dione) were investigated. In vitro, B[a]P
did not displace tritiated 17beta-estradiol ([3H]E2) from either a
bacterially expressed fusion protein consisting of glutathione-S:-

transferase linked to the D, E, and F domains of human ERalpha
(GST-hERalphadef), or from full-length human ERbeta (***hERbeta***) at
concentrations as high as 60 microM. However, 10 microM B[a]P demonstrated
partial agonist activity in human Gal4-ERalphadef and mouse Gal4-ERbetadef
reporter gene assays in transiently transfected MCF-7 cells, relative to
10 nM E2. 1-, 3-, 7-, and 9-hydroxy-B[a]P were found to bind to both
receptor isoforms, each showing a higher affinity for the beta isoform. At
10 microM the four monohydroxylated metabolites were able to induce
Gal4-hERalphadef- and Gal4-mERbetadef-mediated reporter gene expression to
levels 20-100% of that caused by 10 nM E2, suggesting that these
metabolites, and not the parent compound, induced reporter gene expression
following B[a]P treatment of transiently transfected MCF-7 cells. In
addition, the effect of B[a]P on two estrogen-inducible end points,
uterine weight and lactoferrin mRNA levels, was determined in
ovariectomized DBA/2 and C57BL/6 mice. Neither orally administered B[a]P
at doses as high as 10 mg/kg body weight nor subcutaneously injected 3- or
9-hydroxy-B[a]P at doses as high as 20 mg/kg induced effects on uterine
wet weight or uterine lactoferrin mRNA levels in either strain. These data
suggest that B[a]P metabolites that are estrogenic at high concentrations
in vitro do not induce estrogenic effects in the mouse uterus.

L3 ANSWER 5 OF 13 BIOSIS COPYRIGHT 2001 BIOSIS
DUPLICATE 4
ACCESSION NUMBER: 2001:148981 BIOSIS
DOCUMENT NUMBER: PREV200100148981
TITLE: In vitro and in vivo interactions of bisphenol A and its
metabolite, bisphenol A glucuronide, with ***estrogen***
receptors alpha and beta.
AUTHOR(S): Matthews, Jason B.; Twomey, Ken; Zacharewski, Timothy R.
(1)
CORPORATE SOURCE: (1) Department of Biochemistry and Molecular Biology,
Michigan State University, Wilson Road, 223 Biochemistry
Building, East Lansing, MI, 48842-1319: tzachare@pilot.msu.edu USA
SOURCE: Chemical Research in Toxicology, (February, 2001) Vol. 14,
No. 2, pp. 149-157. print.
ISSN: 0893-228X.
DOCUMENT TYPE: Article
LANGUAGE: English
SUMMARY LANGUAGE: English
AB The estrogenic activities of bisphenol A (BPA) and its major metabolite
BPA glucuronide (BPA-G) were assessed in a number of in vitro and in vivo
assays. BPA competed with (3H)-17beta-estradiol (E2) for binding to mouse
uterine cytosol ER, a glutathione S-transferase (GST)-human ER D, E, and F
domain fusion protein (GST-hERalphadef) and full-length recombinant
hERbeta . The IC50 values for E2 were similar for all three
receptor preparations, whereas BPA competed more effectively for binding
to ***hERbeta*** (0.96 muM) than to either mouse uterine cytosol ER
(26 muM) or GST-hERalphadef (36 muM). In contrast, BPA-G did not
competitively displace (3H)E2 from any of the ER preparations. In MCF-7
cells transiently transfected with Gal4-hERalphadef or Gal4-hERbetadef,
BPA induced reporter gene activity with comparable EC50 values (71 and 39
muM, respectively). No significant induction of reporter gene activity was
seen for BPA-G. Cotreatment studies showed that concentrations of (10 muM)
BPA and BPA-G did not antagonize E2-induced luciferase mediated through
either Gal4-hERalphadef or Gal4-hERbetadef. In vivo, the uterotropic
effect of gavage or subcutaneous (sc) administration of 0.002-800 mg of
BPA/kg of body weight/day for three consecutive days was examined in
immature rats. Dose-related estrogenic effects on the rat uterus were
observed at oral doses of 200 and 800 mg/kg and at sc doses of 10, 100,
and 800 mg/kg. These results demonstrate that BPA competes more
effectively for binding to ERbeta, but induces ERalpha- and
ERbeta-mediated gene expression with comparable efficacy. In contrast,
BPA-G did not exhibit any in vitro estrogenic activity. In addition, there
was a clear route dependency on the ability of BPA to induce estrogenic
responses in vivo.

L3 ANSWER 6 OF 13 MEDLINE
ACCESSION NUMBER: 2001129427 MEDLINE
DOCUMENT NUMBER: 21038386 PubMed ID: 11187733
TITLE: Phytoestrogens.
AUTHOR: Kinjo J
CORPORATE SOURCE: Faculty of Pharmaceutical Sciences, Fukuoka University.
SOURCE: NIPPON RINSHO. JAPANESE JOURNAL OF CLINICAL MEDICINE, (2000
Dec) 58 (12) 2434-8. Ref: 20
Journal code: KIM; 0420546. ISSN: 0047-1852.
PUB. COUNTRY: Japan
Journal; Article; (JOURNAL ARTICLE)
General Review; (REVIEW)
(REVIEW, TUTORIAL)
LANGUAGE: Japanese
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200103
ENTRY DATE: Entered STN: 20010404
Last Updated on STN: 20010404
Entered PubMed: 20010122
Entered Medline: 20010301
AB Epidemiological studies revealed that foodstuffs, in particular, soy foods
containing isoflavonoid phytoestrogens may reduce the risk of some
hormone-dependent disease such as not only postmenopausal symptoms but
also certain(breast, prostate and colon) cancers and cardiovascular
disease. This review introduces the metabolism of soybean isoflavonoids by
human intestinal bacteria and the binding and gene-expression activity of
the metabolites towards the human ***estrogen*** ***receptor***
(hER) alpha and beta. The dietary isoflavones(daidzin and genistin) in
soybean were metabolized to equol and dihydrogenistein via daidzein and
genistein, respectively. The metabolites bind more strongly to ***hER***
beta than hER alpha. The binding affinity of genistein is
comparable that of 17 beta-estradiol. Equol induces transcription most
strongly both with ***hER*** ***beta*** and hER alpha.

L3 ANSWER 7 OF 13 CAPLUS COPYRIGHT 2001 ACS
ACCESSION NUMBER: 2000:139632 CAPLUS
DOCUMENT NUMBER: 133:145672
TITLE: Sequencing and cloning of human estrogen .beta.
receptor cDNA in human granulosa
AUTHOR(S): Huang, Hefeng; Mershon, J. L.; Wang, Jinfu
CORPORATE SOURCE: Women's Hospital, School of Medicine, Zhejiang
University, Hangzhou, 310006, Peop. Rep. China
SOURCE: Zhonghua Yixue Zazhi (2000), 80(1), 28-30
CODEN: CHHTAT; ISSN: 0376-2491
PUBLISHER: Zhonghua Yixue Zazhi
DOCUMENT TYPE: Journal
LANGUAGE: Chinese
AB Granulosa cells were prepd. from the ovary of IVF-ET cases by Percoll
technique with Dulbecco's modified eagle medium to analyze the nucleotide
sequence of cDNA and deduce the amino acid sequence of human
estrogen ***receptor*** (***hER*** . ***beta*** .) in
human granulosa cells. RNA was extd. with the TRIzol reagent kit, and
mRNA was purified with oligo-(dT)-cellulose, and cDNA was prepd. from the
mRNA by PCR. Amplified products were cloned into the pGEM-T vector and
transfected into E. coli XL1-Blue. The nucleotide sequence was detd. by
the Sequenase Version 2.0 DNA sequencing kit. The cDNA for ***hER*** .
beta . from human granulosa cells was composed of 1495 bp, contg. a
1431 bp open reading frame, encoding 477 amino acids. The predicted ER
protein included 4 functional domains: A/B, C, D, and E/F. The C domain,
richly contg. cysteine, was the DNA-binding domain (DBD), and E/F domain
was the ligand-binding domain (LBD) among these domains. Detection of
ER.beta. in the ovary granulosa cells played an important role in
explaining the self-endocrine function of estrogen.

L3 ANSWER 8 OF 13 CAPLUS COPYRIGHT 2001 ACS
ACCESSION NUMBER: 1999:127018 CAPLUS
DOCUMENT NUMBER: 130:192325
TITLE: Cloning and cDNA sequence encoding a full-length human
estrogen ***receptor*** -.beta.
INVENTOR(S): Bhat, Ramesh A.; Henderson, Ruth Ann; Hsiao, Chulai;
Karathanasis, Sotirios Konstantinou
PATENT ASSIGNEE(S): American Home Products Corporation, USA
SOURCE: PCT Int. Appl., 49 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 9907847	A1	19990218	WO 1998-US14944	19980720
AU 9884988	A1	19990301	AU 1998-84988	19980720

W: AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CU, CZ, DE, DK, EE, ES, FI, GB, GE, GH, GM, HU, ID, IL, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MD, MG, MK, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, UA, UG, UZ, VN, YU, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM
RW: GH, GM, KE, LS, MW, SD, SZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG
PRIORITY APPLN. INFO.: US 1997-906365 19970805
WO 1998-US14944 19980720
AB The present invention provides isolated nucleic acids encoding full-length
human ***estrogen*** ***receptor*** -.beta. (***hER*** .
beta .), which comprises 530 amino acids. The invention also
provides isolated ***hER*** . ***beta*** . polypeptides and
hER . ***beta*** .-reactive antibodies, including those that
specifically recognize amino acids 1-45 of ***hER*** . ***beta*** .,
which were not previously known. An optimal Kozak translation initiation
sequence is found upstream of the newly discovered initiator methionine
codon, and anal. of expressed transcripts also supports expression of the
long isoform. ***HER*** . ***beta*** . is selectively expressed in
the thymus, spleen, ovary, and testes. In the presence of estradiol,
hERB.beta. long form is about 2-3-fold more active than the short form in
stimulation of estrogen-response elements in HepG2 cells. The invention
also encompasses methods for identifying ***hER*** . ***beta***
.-interactive compds., including agonists, antagonists, and co-activators.
REFERENCE COUNT: 3
REFERENCE(S): (1) Akzo Nobel Nv; EP 0798378 A 1997 CAPLUS
(2) Karobio, A; WO 9709348 A 1997 CAPLUS
(3) Mosselman, S; Febs Letters 1996, V392(1), P49
CAPLUS

L3 ANSWER 9 OF 13 MEDLINE
DUPLICATE 5
ACCESSION NUMBER: 1999174617 MEDLINE
DOCUMENT NUMBER: 99174617 PubMed ID: 10076999
TITLE: ***Estrogen*** ***receptor*** beta activates the
human retinoic acid receptor alpha-1 promoter in

response
to tamoxifen and other ***estrogen*** ***receptor***
antagonists, but not in response to estrogen.
AUTHOR: Zou A; Marschke K B; Arnold K E; Berger E M; Fitzgerald P;
Mais D E; Allegretto E A
CORPORATE SOURCE: Department of Retinoid Research, Ligand Pharmaceuticals,
Inc., San Diego, California 92121, USA.
SOURCE: MOLECULAR ENDOCRINOLOGY, (1999 Mar) 13 (3) 418-30.
Journal code: NGZ; 8801431. ISSN: 0888-8809.
PUB. COUNTRY: United States
Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199905
ENTRY DATE: Entered STN: 19990517
Last Updated on STN: 19990517
Entered Medline: 19990506
AB Human ***estrogen*** ***receptor*** -alpha (hERalpha) or -beta (***hERbeta***) transfected into Hep G2 or COS1 cells each responded to estrogen to increase transcription from an estrogen-responsive element (ERE)-driven reporter vector with similar fold induction through a classical mechanism involving direct receptor binding to DNA. ER antagonists inhibited this estrogen induction through both hERalpha and ***hERbeta*** , although raloxifene was more potent through ERalpha than ERbeta, and tamoxifen was more potent via ERbeta than ERalpha. We have shown previously that estrogen stimulated the human retinoic acid receptor-alpha-1 (hRARalpha-1) promoter through nonclassical EREs by a mechanism that was ERalpha dependent, but that did not involve direct receptor binding to DNA. We show here that in contrast to hERalpha, ***hERbeta*** did not induce reporter activity driven by the hRARalpha-1 promoter in the presence of estrogen. While ***hERbeta*** did not confer estrogen responsiveness on this promoter, it did elicit transcriptional activation in the presence of 4-hydroxytamoxifen (4-OH-Tam). Additionally, this 4-OH-Tam agonist activity via ERbeta was completely blocked by estrogen. Like ERalpha, transcriptional activation of this promoter by ERbeta was not mediated by direct receptor binding to DNA. While hERalpha was shown to act through two estrogen-responsive sequences within the promoter, ***hERbeta*** acted only at the 3'-region, through two Sp1 sites, in response to 4-OH-Tam. Other ER antagonists including raloxifene, ICI-164,384 and ICI-182,780 also acted as agonists through ERbeta via the hRARalpha-1 promoter. Through the use of mutant and chimeric receptors, it was shown that the 4-OH-Tam activity via ERbeta from the hRARalpha-1 promoter in Hep G2 cells required the amino-terminal region of ERbeta, a region that was not necessary for estrogen-induced ERbeta activity from an ERE in Hep G2 cells. Additionally, the progesterone receptor (PR) antagonist RU486 acted as a weak (IC50 >1 microM) antagonist via hERalpha and as a fairly potent (IC50 approximately 200 nM) antagonist via ***hERbeta*** from an ERE-driven reporter in cells that do not express PR. Although RU486 bound only weakly to ERalpha or ERbeta in vitro, it did bind to ERbeta in whole-cell binding assays, and therefore, it is likely metabolized to an ERbeta-interacting compound in the cell. Interestingly, RU486 acted as an agonist through ERbeta to stimulate the hRARalpha-1 promoter in Hep G2 cells. These findings may have ramifications in breast cancer treatment regimens utilizing tamoxifen or other ER antagonists and may explain some of the known estrogenic or antiestrogenic biological actions of RU486.

L3 ANSWER 10 OF 13 MEDLINE
DUPLICATE 6
ACCESSION NUMBER: 1999094591 MEDLINE
DOCUMENT NUMBER: 99094591 PubMed ID: 9879982
TITLE: A novel human ***estrogen*** ***receptor*** beta:
identification and functional analysis of additional N-terminal amino acids.
AUTHOR: Bhat R A; Harnish D C; Stevis P E; Lyttle C R; Komm B S
CORPORATE SOURCE: Women's Health Research Institute, Wyeth-Ayerst Research,
Radnor, PA 19087, USA.. bhatr@war.wyeth.com
SOURCE: JOURNAL OF STEROID BIOCHEMISTRY AND MOLECULAR BIOLOGY,
(1998 Nov) 67 (3) 233-40.
Journal code: AX4; 9015483. ISSN: 0960-0760.
PUB. COUNTRY: ENGLAND: United Kingdom
Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199901
ENTRY DATE: Entered STN: 19990202
Last Updated on STN: 19990202
Entered Medline: 19990121
AB A novel human ***estrogen*** ***receptor*** beta (***hERbeta***) was cloned from human testis mRNA, ovary and thymus cDNA utilizing PCR and 5' RACE methods. The 5' end of ***hERbeta*** contained an additional open reading frame, in-frame and upstream of the published clones. ***hERbeta*** encodes a protein of 530 amino acids with an approximate molecular weight of 63 kDa and is larger than the previously reported rat, mouse and human protein. To determine the functional role of additional N-terminal amino acids, we compared the transcription functions of receptor lacking (hERbetaT) and receptor containing (hERbetaL) this N-terminal extension. hERbetaL is more active than hERbetaT in transactivating ERE-based reporter genes. hERbetaL, but not hERbetaT, attenuated cytokine mediated NFkappaB activation. Taken together, the additional N-terminal amino acids appear to play a role in modulating estrogen responsive gene expression in vitro.

L3 ANSWER 11 OF 13 MEDLINE
DUPLICATE 7
ACCESSION NUMBER: 1998139878 MEDLINE
DOCUMENT NUMBER: 98139878 PubMed ID: 9473491
TITLE: The complete primary structure of human ***estrogen***
receptor beta (***hER*** ***beta***) and
its heterodimerization with ER alpha in vivo and in vitro.
AUTHOR: Ogawa S; Inoue S; Watanabe T; Hiroi H; Orimo A; Hosoi T;
Ouchi Y; Muramatsu M
CORPORATE SOURCE: Department of Biochemistry, Saitama Medical School, Japan.
SOURCE: BIOCHEMICAL AND BIOPHYSICAL RESEARCH COMMUNICATIONS, (1998
Feb 4) 243 (1) 122-6.
Journal code: 9Y8; 0372516. ISSN: 0006-291X.
PUB. COUNTRY: United States
Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
OTHER SOURCE: GENBANK-AB006590
ENTRY MONTH: 199803
ENTRY DATE: Entered STN: 19980326
Last Updated on STN: 20000303
Entered Medline: 19980316
AB Human ***estrogen*** ***receptor*** beta (***hER*** ***beta***) cDNA that encodes the full-length amino acid sequence has been isolated from testis poly(A)+ RNA with the combination of cDNA screening and reverse transcription-PCR. It is composed of a 1590-bp open reading frame and a segment of the 5'- and 3'-untranslated region (UTR) and encodes an additional 53 amino acids in the N-terminal region compared with the previously reported one. Protein interaction between ER alpha and ER beta was demonstrated in vitro by GST pull-down assay and in vivo by immunoprecipitation. Thus, this study indicates that ER alpha and ER beta can interact in vivo, cross-signaling each other.

L3 ANSWER 12 OF 13 MEDLINE
DUPLICATE 8
ACCESSION NUMBER: 1998300286 MEDLINE
DOCUMENT NUMBER: 98300286 PubMed ID: 9636657
TITLE: Cloning and characterization of human ***estrogen***
receptor beta isoforms.
AUTHOR: Moore J T; McKee D D; Slentz-Kesler K; Moore L B; Jones S
A; Horne E L; Su J L; Kliewer S A; Lehmann J M; Willson T M
CORPORATE SOURCE: Department of Molecular Sciences, Glaxo Wellcome Research
and Development, Research Triangle Park, North Carolina
27709, USA.. jtm36008@glaxowellcome.com
SOURCE: BIOCHEMICAL AND BIOPHYSICAL RESEARCH COMMUNICATIONS, (1998
Jun 9) 247 (1) 75-8.
Journal code: 9Y8; 0372516. ISSN: 0006-291X.
PUB. COUNTRY: United States
Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
OTHER SOURCE: GENBANK-AF051427; GENBANK-AF061054; GENBANK-AF061055
ENTRY MONTH: 199807
ENTRY DATE: Entered STN: 19980713
Last Updated on STN: 20000303
Entered Medline: 19980701
AB Multiple transcripts which arise from the human ***estrogen***
receptor beta (ER beta) gene have been characterized. Three full length isoforms of the ***hER*** ***beta*** gene, designated ***hER*** ***beta*** 1-3, were identified in a testis cDNA library. An additional two isoforms, designated ***hER*** ***beta*** 4 and ***hER*** ***beta*** 5, were identified by PCR amplification from testis cDNA and from the MDA-MB 435 cell line. ***hER*** ***beta*** 1 corresponds to the previously described ***hER*** ***beta*** . All five isoforms diverge at a common position within the predicted helix 10 of the ligand binding domain of ***hER*** ***beta*** , with nucleotide sequences consistent with differential exon usage. The ***hER*** ***beta*** isoform mRNAs displayed a differential pattern of expression in human tissues and in tumor cell lines when analyzed by RT-PCR. Further characterization of the three full length isoforms, ***hER*** ***beta*** 1-3, by in vitro band shift studies indicated that the isoforms were able to form DNA-binding homodimers and heterodimers with each other and with the ER alpha subtype.

L3 ANSWER 13 OF 13 MEDLINE
DUPLICATE 9
ACCESSION NUMBER: 97467383 MEDLINE
DOCUMENT NUMBER: 97467383 PubMed ID: 9325313
TITLE: Human ***estrogen*** ***receptor*** beta binds DNA
in a manner similar to and dimerizes with ***estrogen***
receptor alpha.
AUTHOR: Pace P; Taylor J; Suntharalingam S; Coombes R C; Ali S
CORPORATE SOURCE: Department of Medical Oncology, Imperial College of
Medicine, Charing Cross Campus, St. Dunstan's Road, London
W6 8RF, United Kingdom.
SOURCE: JOURNAL OF BIOLOGICAL CHEMISTRY, (1997 Oct 10) 272 (41)
25832-8.
Journal code: HIV; 2985121R. ISSN: 0021-9258.
PUB. COUNTRY: United States
Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199711
ENTRY DATE: Entered STN: 19971224
Last Updated on STN: 19971224
Entered Medline: 19971113
AB The cloning of a novel ***estrogen*** ***receptor*** beta (denoted ERbeta) has recently been described (Kuiper, G. G. J. M., Enmark, E., Pelto-Huikko, M., Nilsson, S., and Gustafsson, J-A. (1996) Proc. Natl. Acad. Sci. U. S. A. 93, 5925-5930 and Mosselman, S., Polman, J. , and Dijkema, R. (1996) FEBS Lett. 392, 49-53). ERbeta is highly homologous to

the "classical" ***estrogen*** ***receptor*** alpha (here referred
to as ERalpha), has been shown to bind estrogens with an affinity similar
to that of ERalpha, and activates expression of reporter genes containing
estrogen response elements in an estrogen-dependent manner. Here we
describe functional studies comparing the DNA binding abilities of human
ERalpha and beta in gel shift assays. We show that DNA binding by ERalpha
and beta are similarly affected by elevated temperature in the absence of
ligand or in the presence of 17beta-estradiol and the partial estrogen
agonist 4-hydroxy-tamoxifen. In the absence of ligand, DNA binding by
ERalpha and beta is rapidly lost at 37 degrees C, while in the presence of
17beta-estradiol and 4-hydroxy-tamoxifen, the loss in DNA binding at
elevated temperature is much more gradual. We show that the loss in DNA
binding is not due to degradation of the receptor proteins. However, while
the complete antagonist ICI 182, 780 does not "protect" human ERalpha
(hERalpha) from loss of DNA binding at elevated temperature in vitro, it
does appear to protect human ERbeta (***hERbeta***), suggestive of
differences in the way ICI 182, 780 acts on hERalpha and beta. We further
report that ERalpha and beta can dimerize with each other, the DNA binding
domain of hERalpha being sufficient for dimerization with ***hERbeta***
. Cell and promoter-specific transcription activation by ERalpha has been
shown to be dependent on the differential action of the N- and C-terminal
transcription activation functions AF-1 and AF-2, respectively. The
existence of a second ***estrogen*** ***receptor*** gene and the
dimerization of ERalpha and beta add greater levels of complexity to
transcription activation in response to estrogens.